

Methodology of Economics

Importance of Good Data Analysis, Methodology, and Theory

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based on work with Koschinsky & Black

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**And Now For Something
Completely Different**

Methodology of Economics

From Heckman & Singer, “Abducting Economics”: *Empirical analyses in economics have diverse goals—all valuable.*

- ① *Some analyses advance knowledge by **uncovering new facts** or providing richer descriptions of old facts.*
- ② *Some seek to **identify causal impacts** of specific interventions as in the literature on **treatment effects**.*
- ③ *Other analyses seek to **understand the mechanisms** producing outcomes with an eye toward interpretation and counterfactual policy evaluation.*

(1) is for a course in Exploratory Data Analysis or Machine Learning

(2) is this course and most econometrics & applied economics

(3) somewhat “orphaned”, the topic of this lecture

“Philosophical” questions:

- What is a **scientific** theory?
- How do we develop a theory?
- How do we test, and either accept or reject a theory?

Who Am I?

Education

- BA physics Harvard
- PhD economics University of Chicago – I sat in your seat, learning econometrics from Heckman, many years ago

Main Career

- Over 20 years in the finance industry
- Trading derivatives, building trading systems, running a hedge fund
- I came back to Chicago 10 years ago, more-or-less by accident

Recently, working on these issues of abduction, theories, and falsification

- With Dan Black, Heckman, others

Outline

① Economic Theories and the “Identification Problem”

“Identification” in Economics – Incomplete View for Developing Knowledge

What is a “Scientific” Theory – A *Fasifiable* Theory

How Do We Develop a Theory – *Abduction*

How Do We Test and Either Accept or Reject a Theory – *Sophisticated Falsification*

② Brief Examples of “Abductive” Economics

A Theory of the Consumption Function and Permanent Income

Heckman’s work on South Carolina

③ Detailed Case Study – John Snow and Cholera in 1850s London

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Overview: John Snow and the Story of Cholera

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Airborne Theories Adopt “Causal Water”

Iterative Process of Scientific Inquiry and Snow’s Theory 1849-66

Falsification and Comparing Theories in 1855

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Cholera Theories and Predictions

Comparing Predictions vs Evidence

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Heckman, James J., and Burton Singer. 2017. "Abducting Economics." *American Economic Review* 107 (5): 298–302.
<https://doi.org/10.1257/aer.p20171118>.

Lays out the case for the iterative process of scientific inquiry, substituting the dynamic interplay between data and theory for the rigid "identification problem" often used in economics

Friedman, Milton. 1953. "Methodology of Positive Economics." In *Essays in Positive Economics*. Chicago: University Of Chicago Press.

A powerful manifesto, as relevant today as 70 years ago, for how to do economics – "a manifesto for abduction" (Heckman)

Lakatos, Imre. 1980. *The Methodology of Scientific Research Programmes: Volume 1: Philosophical Papers*. Edited by John Worrall and Gregory Currie. Vol. 1. Cambridge: Cambridge University Press.

The philosophical foundations for the iterative process of scientific inquiry, building on the work of Popper but with important contributions – particularly Sophisticated Falsification

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“Identification Problem” in Economics

“Identification” in economics is the process of using data to distinguish one model from alternatives

- Incredibly powerful and valuable. But incomplete
- Presumes a class of admissible models

identification analyses take its classes of admissible models as determined before an empirical investigation begins. (Heckman & Singer 299)

- But as Friedman points out, the class of models (hypotheses) is actually infinite:

Observed facts are necessarily finite in number; possible hypotheses, infinite. (Friedman 9)

identification analyses take its classes of admissible models as determined before an empirical investigation begins. ... The rigid separation of the processes of model generation and model testing -- a central feature of the formulation of the identification problem -- while analytically convenient -- is artificial (Heckman & Singer 299)

- Friedman’s fn 11 is particularly cogent in describing it’s origin and outline

“Identification” as Incomplete for Developing Knowledge

Challenging the standard identification framework is heresy, but it must be done:

The abductive mode of thought challenges the currently influential framework of the “identification problem,” which underlies both treatment effect and structural approaches ... The rigid separation of the processes of model generation and model testing -- a central feature of the formulation of the identification problem -- while analytically convenient -- is artificial

It is not only artificial, but it does not work and is violated by many good practicing scientists, who regularly go back-and-forth between data and theories:

Fortunately [this rigid separation is] usually ignored by more seasoned empirical economists.

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What is a “Scientific” Theory – Falsifiability

- Commonly termed the “demarcation” problem – demarcating science from pseudo-science
- Popper’s solution: falsifiability: hypotheses or theories or statements that make falsifiable predictions.
 - Need to provide some circumstances under which we would be willing to abandon our theory or hypothesis. Science can be refuted by the facts, pseudo-science cannot
- This leads to *Naive Falsification* – specify (up-front) a set of observations which, if observed, would lead one to abandon the theory

Naive Falsifiability Unworkable

- But this turns out to be fiendishly difficult, even impossible
- Contrary observations (“anomalies”) can always be rationalized:

Nature may shout no, but human ingenuity ... may always be able to shout louder. With sufficient resourcefulness and some luck, any theory can be defended 'progressively' for a long time, even if it is false. (Lakatos 111)

The direction of science is determined primarily by human creative imagination and not by the universe of facts which surrounds us. Creative imagination is likely to find corroborating novel evidence even for the most 'absurd' programme, if the search has sufficient drive. (Lakatos 99)

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New Knowledge (Theory) – Not from Deduction or Induction

Deduction: deriving new true statements from earlier (true) statements.

- Deduction only ensures the reliable *transmission* of truth or knowledge, and cannot by itself *increase* or create truth-content.

Induction: generalizing from evidence.

- The “problem of induction” going back to Hume.
- We cannot use a set of finite observations – say “all swans so far observed are white” – to generalize to a theory about the world – say “All swans are white”.

Observed facts are necessarily finite in number; possible hypotheses, infinite. If there is one hypothesis that is consistent with the available evidence, there are always an infinite number that are.” (Friedman 9)

Abduction or “Inference to the Best Explanation”

Peirce's *Abduction*:

The surprising fact, C, is observed;

But if A were true, C would be a matter of course

Hence, there is reason to suspect that A is true. (CP 5. 189, 1903; see also 2. 624, 1878.) (quoted in ?, 93-94)

Two key elements here

- ① We can *expand* beyond our existing knowledge (circumventing the problems with deduction)
- ② We *suspect* but do not know that it is true (circumventing the problem with induction from data)

More Broadly – Iterative Process of Scientific Inquiry

Scientific inquiry and the growth of knowledge is an ambiguous, uncertain, complex process.

- Not progressing mechanically, difficult to quantify
- A complicated and dynamic interplay between data, theory, and testing

We call upon the work of two (three) philosophers

- Charles Sanders Peirce (1839-1914), “father of pragmatism”, he proposed a “path of inquiry”
 - Three stages of scientific inquiry: *Abduction, Deduction, Induction*
- Imre Lakatos (1922-1974), philosopher of science, student of Karl Popper
 - “Unit of appraisal” for scientific inquiry is a *research programme* – collection of theories and hypotheses with structure
 - *Sophisticated Falsification* for comparing and deciding between programmes



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Falsification: Dogmatic, Naive, Sophisticated

Karl Popper (1902-1994) introduced idea of *Falsification* and *Falsifiability*

- Trying to solve a problem: We cannot *Verify* a scientific theory. But surely we can *Falsify* it?
- Science: theories that could be falsified or refuted

Turns out Falsification is not so simple

- A contradiction implies some particular *hypothesis* or theory under scrutiny, surrounded by (and tested using) accepted facts and theories
- But any “accepted” fact or theory is only accepted provisionally, always subject to revision (Peirce recognized this)
- Instead of rejecting the *hypothesis*, we may revise the “facts” or “accepted theories” to make the contradiction go away.
- Essence of the Duhem-Quine thesis

Research Programmes and Sophisticated Falsification

Scientific Research Programme as the fundamental unit we work with:

- Not an isolated hypothesis, but a developing series of theories
- *Hard core* – not (generally) subject to revision or refutation
- *Auxiliary belt* – translate core to world of observations, readily revised, added

Sophisticated Falsification: a scientific theory T is falsified if and only if another theory T' has been proposed for which:

- ① T' has excess empirical content (predicts novel facts, not predicted by T)
- ② T' explains previous success of T (unrefuted content of T is included)
- ③ Some of the excess content of T' is corroborated

Not really *falsification* at all, but a ***criterion for supplanting***

Progressive versus Degenerating Programmes

Adjustments and additions to hypotheses and theories – generally auxiliary belt – allowed

- **Progressive**: generate *new* predictions and new facts
- **Degenerating**: remove and account for anomalies, but do not generate new facts or theoretical insights

This distinction is the essence of Lakatos's methodology, essence of supplanting an old theory with new

Lakatos's conjecture (and I do think we need to treat it as a conjecture) is that *Progressive* programmes lead to increases in knowledge, *Degenerating* programmes do not.

- Foundational problems in defining and talking about *knowledge* and *truth* mean that I think this is a conjecture. But a very useful one.

Iterative Process of Scientific Inquiry

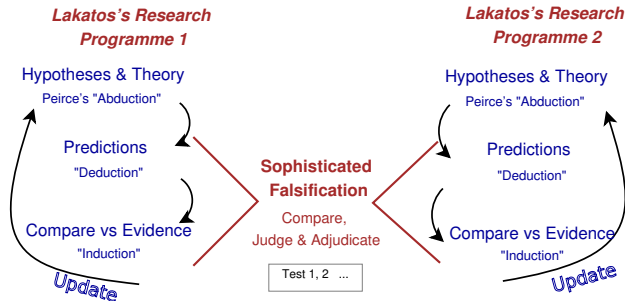
Peirce's 3 stages of Inquiry

- 1 Develop & modify theories (abduction)
- 2 Generate predictions from theories (deduction)
- 3 Test predictions vs evidence (induction)

On-going process

Research Programme as unit of analysis from Lakatos. We also need a *criterion* for choosing between theories and programmes – when is one theory thrown out and supplanted by another

- Something more than Thomas Kuhn's psycho-social *Scientific Revolutions*
- Popper's proposal of *falsification* seems like the answer, but it does not work – the how and why holds the answer to airborne theories' adoption of water







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Permanent Income and *Theory of the Consumption Function*

Puzzle, related to Keynes's *General Theory*, Marginal Propensity to Consume, and fiscal multiplier

- MPC: How much does Cons \uparrow when Inc \uparrow ? How much spent vs saved?
- Cross-section (point-in-time): MPC low, most saved (savings \uparrow)
- Time-series & cross-country: MPC near one, savings rate constant

Friedman has wonderful, and wonderfully simple, explanation:

- Permanent vs Transitory Income: $\Delta Y = \Delta Y_{perm} + \Delta Y_{trans}$
- $MPC_{perm} \approx 1$, $MPC_{trans} \approx 0$
- We often mis-measure “income”:
 - Measure across people, much income difference Y_{trans}
 - For aggregate (measure across time) Y_{trans} averages out, see ΔY_{perm}
- Friedman's 7-day week example: On Wed, 6 workers earn \$0, 1 earns \$100 and saves most (spends little, MPC low)

Hugely relevant for today's questions about government tax and spending stimulus

- Multiple strands of evidence

Diverse data were analyzed and differences reconciled using and extending basic economic theory. Not a single p-value is reported in what many consider one of the most influential empirical studies in the history of economics. Instead, it reports a running dialogue with data, models and with new models that emerged from his immersion in a vast array of data. (Heckman 300-301)

- Generating new (testable) predictions (regression of income on consumption, distribution of income and wealth)

The hypothesis has many empirical implications in addition to those already stated about the regression of measured consumption on measured income [the empirical implications that initiated the hypothesis] (Friedman p 224)

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Heckman & Payner – Civil Rights act was “Causal”

- Two decades of research following the Civil Rights Act of 1964 failed to produce professional consensus on the contribution of federal government civil rights activity to the economic progress of black Americans.
- Heckman & Payner addressed this question by using empirical proof by elimination.
 - Using a variety of data sources and measures of federal activity and eliminating other plausible explanations, conclude that federal policy benefited black economic status in South Carolina.

They did not start out believing it was the Civil Rights Act

- No “causal identification strategy”
- Basically, they tried everything, until they (and their audience) could come to no other conclusion except “the Civil Rights Act was causal”

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What Causes Cholera? Hugely Important in 1850s London



Horrendous way to die – dehydration, convulsions, blue skin, die within hours

Scourge of mid-1800s London – 1831-32 6,526 dead; 1849 14,137; 1853-54 10,738
Massive uncertainty as to cause

- Bad air (miasma); “bad breeding” (poverty); bad ground (plague pits)

Huge public health question – one man knew the answer, but nobody listened:

- John Snow & fecal-oral transmission – effort to prove causal theory

Rational Reconstruction of History: How Science *Is Done*

Snow, in 1849 and again 1855, provided strong evidence – ***but failed to convince public & medical health establishment!***

- Current-day discussions center around “water as causal effect”
- *Treatment Effect* approach to causality (potential outcome framework, what Pinto&Heckman call “effects of causes”)
- Snow credited with first use of difference-in-differences & randomization as IV

We undertake *rational reconstruction* of competition among theories in 1850s

- Dynamic interplay of theory & data (Peirce, Lakatos, Heckman & Singer)
- Snow used multiple strands of evidence (some statistical, some not)

Helps us understand

- Why fecal-oral theory superior
- How alternatives (rationally) survived
- How to demonstrate a causal explanation

Recognize the Treatment Effects view of causality is insufficient for building a causal explanation (understanding the causes of cholera)

Snow as Example: How Science *Should Be Done*

Common paradigm for empirical social science is static:

First *a priori theory* Then **statistical testing**

Snow teaches us that scientific inquiry is a dynamic & iterative process

- Following Peirce and Lakatos, examine 1850s competition among theories

Iterative process of inquiry

- 1) Theory; 2) Predict; 3) Compare; 4) Update

Sophisticated Falsification

- Criterion for comparing research programmes

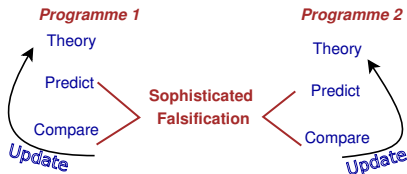
Update & Modify Theories

- Demonstrates both **Abduction** and **Protective Auxiliary Hypotheses**

Snow as example / case study – template for how to do science

- Explicitly build out Sophisticated Falsification: *Predict & Compare*
- Historical examination of evidence, and template for how to do science

Focus on *observational* (aggregate, epidemiological) rather than *biological* (experimental) evidence



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Cholera – Disease of Poor Sanitation

What is Cholera?

- *Vibrio Cholerae* – bacterium that infects the small intestine of humans
- Causes severe diarrhea (& vomiting) that drains fluids
- Death from dehydration & organ failure
- Oral Rehydration Therapy highly successful (roughly 1960s)
 - In case you ever need it, here's the recipe – 1 liter boiled water, 1/2 teaspoon salt, 6 teaspoons sugar, mashed banana (potassium)

Cholera thrives in crowded cities with poor sanitation

- Transmitted through (inadvertent) ingestion of fecal matter
- When cholera exits one victim, needs to find a way into gut of others
- Commonly contaminated water – recycling (drinking) sewage
- Victorian London was an ideal playground for cholera to thrive

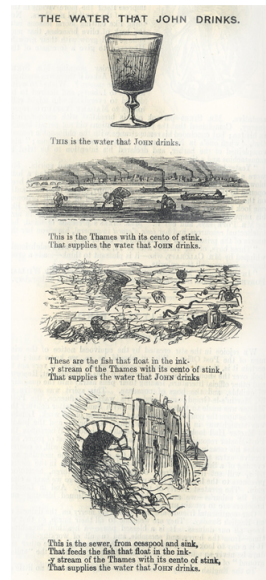
Cholera Loved Victorian London

Victorian London was an ideal playground for cholera

- Mid-1800s London was dirty, smelly place with no organized sewage treatment
- Efforts to improve sanitation made things worse
 - cesspools relatively safe – did not provide access to thousands of guts
- Public Health Act of 1848 required houses to connect to sewage lines
 - helped clean up streets, flushed filth to Thames
- By mid-1800s, cholera had easy access from the gut of one to thousands of victims

Contemporaries were aware of dirty water (*Punch* 1849)

- But water not recognized as vector for cholera



Solution – Construction of Bazalgette “Outfall Sewers”

Sewers that sloped towards outfalls (discharge points) lower on the Thames

- Construction started (under Bazalgette) 1859, response to 1858 “Great Stink”
- Embankments along Thames – what we see today
 - Embedded discharge pipes – still used today (?)
 - Decreased width, increased flow – scouring effect
- Moved sewage downstream, below London & water in-take



One final outbreak, 1866, limited to east London, last area unserved by sewers

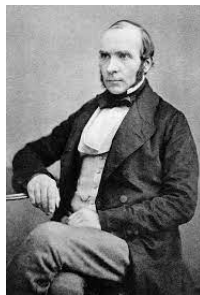
John Snow's Research & Publications

Doctor – pioneer in anesthesia & medical hygiene

- Provided Queen Victoria with anesthesia during childbirth

Research and writing on Cholera

- 1849: “On the Mode of Communication of Cholera”
 - Laid out theory and evidence for waterborne transmission
- 1855: “On the Mode of Communication of Cholera”
 - Substantially expanded, additional evidence and argument (DiD & randomization)
- 1856: “Cholera and the water supply in the south district of London in 1854”
 - “Actual vs predicted” for other causes of cholera



John Snow's 1849 Theory & 1855 Evidence

1849: Snow developed theory of infection & transmission

- Based on medical knowledge and study of single events – Horsleydown & Albion Terrace

Fully-developed & modern theory of disease

- Infects & reproduces in the small intestine
- Exits from victim, another through contact or water

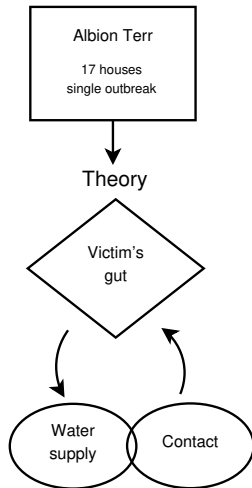
Implications for patterns of infection, across scales

- Person-to-person (normal)
- Neighborhood (localized water, explosive)
- Municipal (drinking water, widespread)

Snow's work grounded by theory

Snow had a good idea – a causal theory about how the disease spread – that guided the gathering and assessment of evidence. (Tufte)

1855: evidence & argument to convince skeptics – effort at *Falsification*



Alternative Theories – Airborne (Inhaled)

For our purposes – predicting cholera observations – alternatives were **Airborne**

- One version was **Miasma** – general atmospheric influence
- For all, cholera poison was airborne and (generally) inhaled

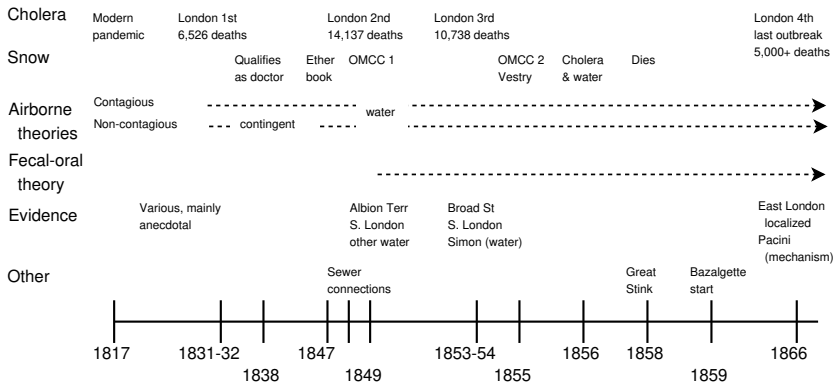
Important debates, *which we can largely ignore* – **airborne** is important

- Contagious: transmitted person-to-person
- Non-contagious: atmospheric, general or localized environmental factors
- Contingent-contagion: introduced 1830s due to contradictory observations
- Localization: non-contagious, specific local factors (e.g. dampness)

All theories posited **predisposing causes** and **susceptibilities**

- Crowding, poverty, dampness, filth (sewage, smells), graveyards
- None absolutely crazy – often correlated with cholera (and dirty water)
- Elevation important (empirically and historically – Farr)

Timeline – For Events, Snow, Theories, Data



1858 – Snow’s theory not widely accepted – his Lancet obituary, no mention of cholera

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Standard Approach – “Causal Water” & Treatment Effects

1850s – Strong evidence supporting water as causal

- John Snow, but many others (John Simon, John Sutherland, Rev. Henry Whitehead, William Farr) demonstrated strong evidence

They did not have statistical tools, but recognized causality issues

- Snow used a nascent difference-in-differences, Simon recognizable DiD.
- Snow used randomization as IV
- Discussion of effect and importance of randomization by Farr (and Snow) is quite modern

Seems clear-cut case of “Falsification” & “Refutation”

- Airborne theories predict infection by breathing
- Fecal-oral theory predicts infection by drinking contaminated water

Yet “causal water” did not move medical establishment to fecal-oral theory

- Presented as example of “smart people cling[ing] to an outlandishly incorrect idea despite substantial evidence to the contrary” (Johnson)

Snow's "Grand Experiment" – Water Supply Changes

Two water companies served south London – Southwark & Vauxhall Co and Lambeth Co. – 486,936 customers, 300,000 **intimately mixed**

- In 1830s & 1840s companies competed for customers, often on same street
In many cases a single house has a supply different from that on either side. Each company supplies both rich and poor, both large houses and small; there is no difference in the condition or occupation of the persons receiving the water of the different companies. (Snow 1855 p 75)

1849 epidemic

- Both companies drew water from low in the Thames – near Vauxhall bridge

1852

- Lambeth Company moved source to Thames Ditton (upstream of London)
- In response to Act of Parliament, requiring move (by 1855)

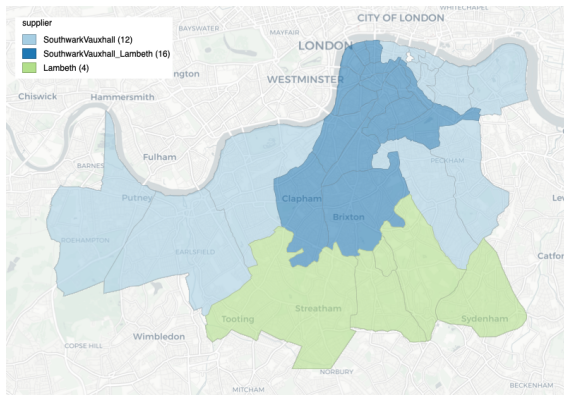
1854 epidemic

- Southwark & Vauxhall Co supplied dirty water
- Lambeth Co supplied cleaner water

32 Subdistricts, 12 S&V only, 16 joint, 4 Lambeth

Registration Districts & Sub-Districts – Need to keep straight

- Deaths collected weekly by Registrar-General, by District & Subdistrict
- In this region of South London, 32 sub-districts
- Some supplied S&V only, others joint
- DiD: compare “S&V only” vs “joint”
- Mixing & randomization: ideally, compare within “joint”
- “First 12” (light blue) – Southwark & Vauxhall Water Co only – dirty water 1849 & 1854
- “Next 16” Mixed or Joint (dark blue) – Southwark & Vauxhall Co *and* Lambeth Water Co – 1849 dirty water, 1854 part dirty (S&V) & part clean (Lambeth)
- “Final 4” (green) – Lambeth Water Co only – not relevant, not supplied in 1849



Deaths: Combined (All Suppliers) vs Direct (By Supplier)

Data available in 1855

- Deaths (combined all suppliers) 1849 & 1854, full epidemic
- Population (combined all suppliers)
- Deaths by supplier, first 7 weeks of epidemic (collected by Snow)

Data available in 1856 (originally published by Simon)

- Population by supplier (only S&V shown here)

	subdistricts	Deaths 1849	Deaths 1854	Supplier	Population 1851	1854, first 7 wks		Pop S&V
						Deaths S&V	Deaths Lam	
1	St. Saviour	283	371	SV	19,709	115	0	16,337
2	St. Olave	157	161	SV	8,015	43	0	8,745
13	Christchurch	256	113	SV & Lambeth	16,022	11	13	2,915
14	Kent Road	267	174	SV & Lambeth	18,126	52	5	12,630
29	Norwood	2	10	Lambeth	3,977	0	2	0
	TOTAL	6,328	5,042		486,936	1,263	98	266,516

Combined (all suppliers)

Direct (by supplier)

$$D_{subdist} = D_{S\&V} + D_{Lam} + D_{Other}$$

$$\{D_{S\&V}, D_{Lam}, D_{Other}\}$$

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Snow's Analysis – Two Approaches

Mixing or quasi-random direct comparison – Randomization is IV

- Snow determined supplier – by bill or chloride test
- Visited all houses (deaths) for 7 weeks ending Aug 26

Diff-in-Diffs comparison of combined (all suppliers) mortality rates

- For each subdistrict, observe combined deaths all suppliers
- Compare 1849 vs 1854 and Treated (clean) vs untreated (dirty) subdistricts

Snow Modern in View of Mixing (Randomization)

Recognized that mixing (randomization) would average out differences

As there is no difference whatever, either in the houses or the people receiving the supply of the two Water Companies, or in any of the physical conditions with which they are surrounded, it is obvious that no experiment could have been devised which would' more thoroughly test the effect of water supply on the progress of cholera than this. (1855 p. 75)

Cited as first instance of Randomization and Instrumental Variables (Greene 2018, also Deaton, others)

Comparison of Mixed or Randomized Population

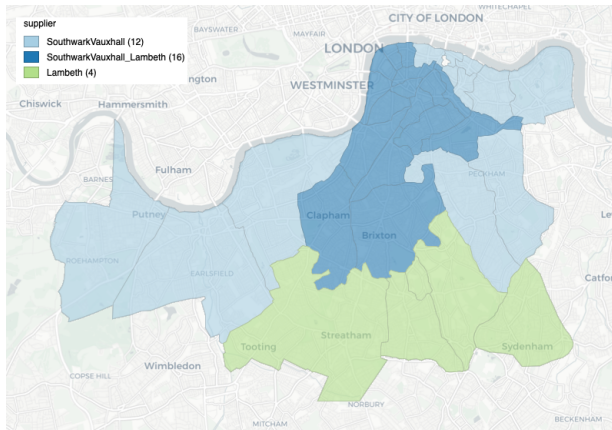
Table: Houses, Deaths, and Mortality Rates per 10,000 Households, First Seven Weeks of 1854 Cholera Epidemic – Table IX

Water Supplier	Number of houses	Deaths from Cholera	Deaths in each 10,000 houses
Southwark & Vauxhall Co supply	40,046	1,263	315.4
Lambeth Co supply	26,107	98	37.5
Rest of London	256,423	1,422	59
Ratio Effect: Southwark & Vauxhall vs Lambeth			8.40

Note that this corrects a rounding error in the “Deaths in each 10,000 houses” for Lambeth in Snow’s original table

- Found LARGE Lambeth effect
- But suffered from potential confounding – includes **all** subdistricts

Problem: Snow's Mixing Comparison Uses *All* Subdistricts



- Snow wanted to limit analysis to Joint (Mixed) subdistricts – could not
- Population (houses) by supplier for overall region only
- Potential for confounding (bias if S&V-only subdistricts different than joint)

Second Approach – DiD – Before v After, Treated v Control

Comparing the S&V-only subdistricts vs the Jointly-supplied subdistricts

- Interestingly, Snow did not convert deaths to rates – missed an opportunity
- Large treatment effect, but need to evaluate statistical significance

Mortality Rates 1849 & 1854, Summary Snow 1855 Table XII

	1849 Deaths per 10,000	1854 Deaths per 10,000	Ratio 1849 - 1854
Always Dirty – Southwark & Vauxhall Water Company Only (“First 12” subdistricts)	134.9 dirty, S&V only	146.6 dirty, S&V only	0.92 diff in time
Dirty / Clean – Joint Southwark & Vauxhall and Lambeth Companies (“Next 16” subdistricts)	130.1 dirty, joint	84.9 (partial) clean	1.53 diff in time & treatment
Ratio: Next 16 less First 12	0.96 diff in region	1.73 diff in region & treatment	1.67 (partial) treatment

Problem: treatment effect only marginally significant

DiD as Regression

$$\ln(\text{Rate}_{\text{subdist},\text{yr}}) = \ln(\text{count}_{\text{subdist},\text{yr}} / \text{population}_{\text{subdist},\text{yr}}) = \hat{\mu} + \hat{\delta}_{54} \cdot I_{\text{yr}=1854} \\ + \hat{\gamma}_J \cdot I_{\text{subdist}=\text{joint}} + \hat{\beta} \cdot I_{\text{subdist}=\text{joint}} \cdot I_{\text{yr}=1854} + \varepsilon_{s,\text{yr}}$$

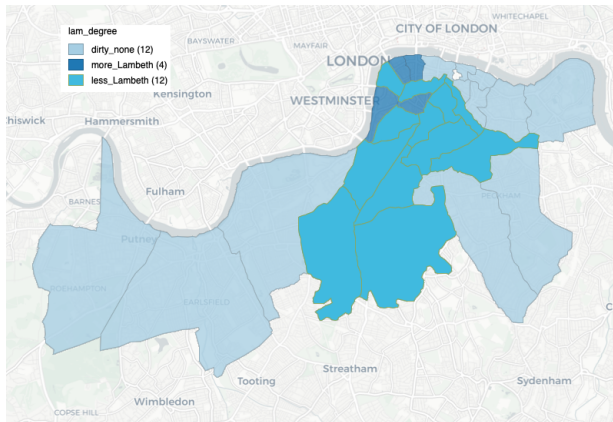
Region or Sub-Districts – Supplied by	1849 Death Rate (log)	1854 Death Rate (log)	Diff 1854 less 1849
First 12 – Southwark Only	μ	$\mu + \delta_{54}$	δ_{54}
Next 16 – Joint Southwark and Lambeth	$\mu + \gamma_J$	$\mu + \delta_{54} + \beta + \gamma_J$	$\delta_{54} + \beta$
Diff Joint less Southwark	γ_J	$\beta + \gamma_J$	β

Regression framework allows us to

- Use subdistrict detail, and additional regressors (if available)
- Test for statistical significance (both for finite population and “within-sample” variation)
- Extend the DiD framework to continuous treatment and actual-vs-predicted

NOTE: some important econometric issues here – need to use count (Poisson or Negative Binomial) regression – Generalized Linear Models

Snow Highlighted Difference in “Lambeth Degree”



- Four subdistricts where “the supply of the Lambeth Water Company is more general than elsewhere”

Snow Highlighted Difference in “Lambeth Degree”

	1849 Deaths per 10,000	1854 Deaths per 10,000	Ratio 1849 - 1854
Always Dirty – Southwark & Vauxhall Water Company Only (“First 12” subdistricts)	134.9 dirty, S&V only	146.6 dirty, S&V only	0.92 diff in time
Dirty / Clean – “More Lambeth” in Joint (4 subdistricts)	138.8 dirty, more	47.2 more clean	2.94 time & more
Dirty / Clean – “Less Lambeth” in Joint (12 subdistricts)	127.6 dirty, less	95.6 less clean	1.34 time & less
Ratio: “More Lambeth” vs Dirty	0.97 diff in region	3.11 region & more	3.20 more treatment
Ratio: “Less Lambeth” vs Dirty	1.06 diff in region	1.53 region & less	1.45 less treatment

Larger effect for “More Lambeth”

- Now, treatment effect is highly significant (see below)

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Many Contributed Evidence for “Causal Water”

1849:

John Sutherland – Board of Health official

- 1849, Hope Street, Manchester, 90 houses, 25 deaths.
- 30 used pump water, 25 deaths; 60 used other water, 0 deaths

William Farr – head of statistics, General Register Office

- “Dr. Snow is unfortunately able to show that this excremental distribution [waterborne] ... is possible to a very considerable extent”
- Table (1853 publication) showing deaths in 1849: “impurity of the waters .. is in nearly a direct proportion to the mortality from cholera”

1854

John Simon Medical (Officer of Health for the City of London)

- DiD (more explicit than Snow’s): “final solution of any existing uncertainty as to the dangerousness of putrefiable drinking-water”

Rev. Henry Whitehead (working on Broad St, ultimately ally of Snow’s)

- Those who drank vs did not drink – essentially 2x2 contingency table

“Causal Water” Wonderful, But Useless

Snow’s work wonderful example of causal analysis

- Good for teaching – simple data, important social problem, valuable techniques (DiD & randomization), clean results

But proving water was causal had little impact – fecal-oral theory not widely accepted in 1850s.

Why?

Need deeper view of scientific inquiry than treatment effects

- We are not minimizing importance or value of treatment effects framework
- It is a crucial component – but only a component – of overall iterative scientific inquiry

Airborne Theories Adopt “Causal Water”

Alternatives – Airborne Theories – adopted water as a contributing cause of cholera

Understanding *The Iterative Process of Scientific Inquiry* shows why this was unfortunate, but not irrational

- Not *necessarily* a case of “smart people cling[ing] to an outlandishly incorrect idea despite substantial evidence to the contrary” (Johnson)

Remember Iterative Process of Scientific Inquiry

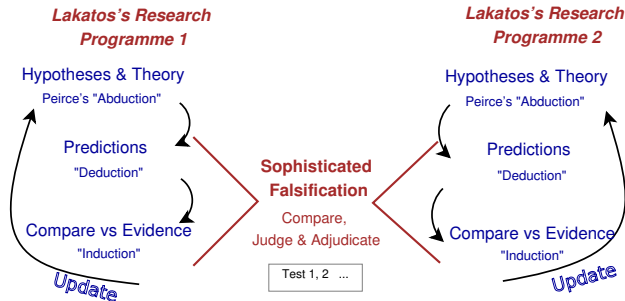
Peirce's 3 stages of Inquiry

- 1 Develop & modify theories (abduction)
- 2 Generate predictions from theories (deduction)
- 3 Test predictions vs evidence (induction)

On-going process

Research Programme as unit of analysis from Lakatos. We also need a *criterion* for choosing between theories and programmes – when is one theory thrown out and supplanted by another

- Something more than Thomas Kuhn's psycho-social *Scientific Revolutions*
- Popper's proposal of *falsification* seems like the answer, but it does not work – the how and why holds the answer to airborne theories' adoption of water



Cholera 1849-1866 as an Example of the Iterative Process

Apply ideas of Peirce and Lakatos to developments 1849-1866

1849, Snow's abductive leap

- Surprising fact *C* (anomaly): airborne cholera seems sometimes contagious, sometimes not
- If hypothesis *A* (intestinal, fecal-oral transmission) were true, *C* would be a matter of course

OMCC: Generating predictions, testing against evidence

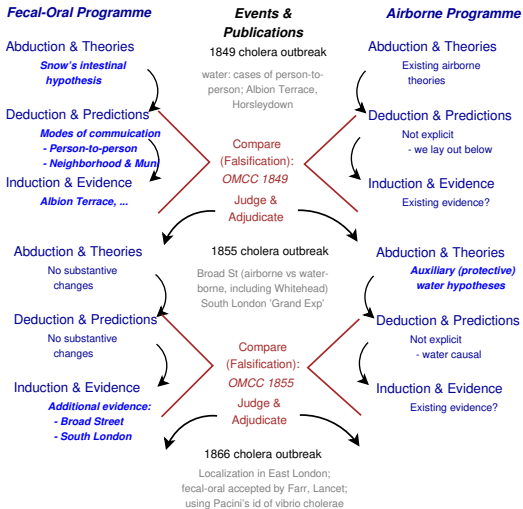
- Effort at *Falsification* – limited acceptance

Airborne response

- New auxiliary hypotheses: water as causal, water transmission

1855: new evidence, new round

1866: new evidence wider acceptance



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1855 Comparison and Falsification

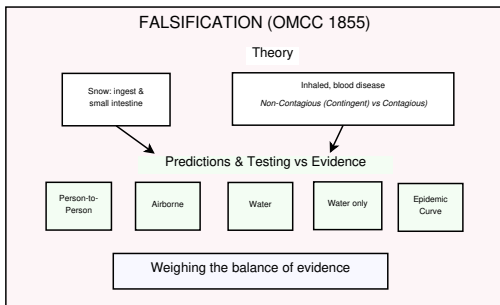
We view Snow's 1855 *On the mode of communication of cholera* as an extended effort at falsification – demonstrating the superiority of the fecal-oral to alternative theories

Steps for falsification:

- 1 Lay out competing theories
- 2 Develop predictions from theories
- 3 Compare predictions versus evidence

Echos approach of Katz & Singer

- Assemble broad range of disparate evidence, varying forms and quality



Here, hypothesis testing in a treatment effects framework takes the role of strengthening the weight of (some) evidence

- For example, by reliably showing that water is *causal*, and observed association is not spurious (causation and not correlation)

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Cholera Theories

Snow (1849, 1855) carefully laid out his theory. For others – we have read contemporary accounts (and learned from Vinten-Johansen, noted Snow scholar)

Primary Alternatives – Airborne

- Variety of theories, contagious vs non-contagious
- Thought to act on the blood
- Wrt predictions, “airborne cholera poison” is important characteristic

Intestinal

- Snow’s fecal-oral contagious primary focus
- Budd’s non-contagious intestinal minor

	Infection (contagious): Poison produced in victims’ bodies, person-to-person
Blood Disease, Airborne (Morbid poison in atmosphere, transmitted via inhalation, with water possible post-1849)	General Atmospheric (generally non-contagious) Poison in atmosphere (not person-to-person)
	Localization (generally non-contagious): Localized sources organic decomposition (eg sewer gases)
	Elevation / Zymotic: A refinement of localization (above).
Gastrointestinal tract, Ingested	Alimentary Canal (contagious, now termed fecal-oral transmission): “excretions of the sick ... being accidentally swallowed” (Snow 1849)
	Intestinal Canal (non-contagious) – Budd

Primary Distinction – Transmission: Fecal-oral vs Airborne

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- Contagion: contradictory airborne predictions were (abductive) source of Snow's theory
- Divergent predictions ⇒ discriminating between theories – high value
- Transmission by air subtle implication of Snow & Whitehead (Broad St)
- Auxiliary water hypotheses change airborne predictions
- Epidemic curve (time patterns) *new empirical content* of Snow's theory – corroborated

More predictions in paper

	Prediction/Observation	Predict? Fecal	Air	Value
PERSON-TO-PERSON CONTAGION				
2a	Airborne: only between those in close proximity, sharing airspace	N	Y	High
2b	Non-airborne: those in close proximity, via sharing food, clothing	Y	N	
GENERAL CHARACTERISTICS				
4	<i>Transmission by air:</i> Sharing air (not water) ⇒ similar mortality; not sharing air (same water) ⇒ different mortality	N	Y/N	Medium
5b	Water is causal: Airborne theories change pre-vs-post-1849	Y	N/Y	Low
5c-f	Other factors: variety of factors, independent of water, eg Overcrowding & poor ventilation	N	Y	Medium
EPIDEMIC CURVE				
8	Neighborhood: Mortality grows quickly then falls off	Y	N	High
9	Municipal: Early: large difference by exposure. Later: difference diminishes	Y	N	High

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Predictions versus Evidence – For *Sophisticated Falsification*

"P/N" indicates protection from auxiliary hypothesis

- Oral-fecal predictions make contagion evidence consistent – supports fecal, contradicts airborne
- Water vs air transmission no longer distinguishes – air theories "protected"
- "Other factors" crucial, new prediction, separates theories.
- Snow recognized importance, others less-so
- Snow lacked statistical tools for definitive conclusion
- Time-pattern (*epidemic curve*) also new, also separates theories

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2b	sometimes not. Anecdotes, reports, and case studies; eg Blane & Corbyn, Snow			
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5b	<i>Water causal:</i> Narrative: neighborhoods (Albion ...); quantitative: municipal & nbhd (Snow, Sutherland, Simon)	Y	P/N	Low
5c-f	<i>Other factors:</i> Snow (1856) "overwhelming influence" of water; modestly successful (no statistics)	Y	N?	Medium
EPIDEMIC CURVE				
8	<i>Neighborhood:</i> Narratives, growth explosive from low backg'd (Snow)	Y	N	High
9	<i>Municipal:</i> Snow highlighted (1855, 1856a, 1856b, 1857)	Y	N	High

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Airborne Theories Predict Transmission by Air

- ① Strong evidence that in Broad St cholera *not* transmitted via air
 - Does *not* rule out that it *could* be transmitted via air in other circumstances
 - But lack of evidence for airborne transmission weakens airborne theories
- ② Strong evidence that in Broad St cholera *is* transmitted via water
 - Does not refute airborne theories
 - Protective auxiliary hypotheses allow water as either predisposing factor (say reduced immunity) or mode of transmitting airborne cholera poison

Prediction

- Sharing same air (different water) \Rightarrow similar mortality
- Sharing different air (same water) \Rightarrow different mortality

Evidence

- Snow & Broad St: Workhouse & brewery same air as nearby buildings, much lower mortality (separate wells)
- Whitehead & Broad St: collected data allowing 2x2 contingency analysis
- Snow & Broad St: Widow Eley in Hampstead, sons sent water, she died
- Whitehead & Broad St: collected data allowing 2x2 contingency analysis

Multiple strands of strong evidence that water *causes* cholera

- Narratives of neighborhood outbreaks (Manchester, Albion Terrace, Horsleydown 1849)
- Snow's difference-in-differences & randomized comparison
 - Modern re-analysis reinforces the strength of Snow's evidence
- Simon's improved randomized comparison

But – Auxiliary hypotheses protected airborne theories

- Water as predisposing cause – e.g. contaminated water could reduce natural immunity or contribute to decay & production of airborne cholera poison
- Water as mode of transmission – airborne poison dissolves in water, could even be transmitted through ingestion (stomach & intestines)

Causal water was no longer useful for distinguishing between theories

Predictions versus Evidence – For *Sophisticated Falsification*

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PERSON-TO-PERSON CONTAGION				
2a	in some cases those sharing the same airspace are infected,	Y	N	High
2b	sometimes not. Anecdotes, reports, and case studies; eg Blane & Corbyn, Snow			
GENERAL CHARACTERISTICS				
4	Transmission by air: Broad Street events, data, sharing air not water, vice-versa (Snow, Whitehead)	Y	P/N	Medium
5b	Water causal: Narrative: neighborhoods (Albion ...); quantitative: municipal & nbhd (Snow, Sutherland, Simon)	Y	P/N	Low
5c-f	Other factors: Snow (1856) “overwhelming influence” of water; modestly successful (no statistics)	Y	N?	Medium
EPIDEMIC CURVE				
8	Neighborhood: Narratives, growth explosive from low backg'd (Snow)	Y	N	High
9	Municipal: Snow highlighted (1855, 1856a, 1856b, 1857)	Y	N	High

Predictions versus Evidence – For *Sophisticated Falsification*

"P/N" indicates protection from auxiliary hypothesis

- Oral-fecal predictions make contagion evidence consistent – supports fecal, contradicts airborne
- Water vs air transmission no longer distinguishes – air theories "protected"
- "Other factors" crucial, new prediction, separates theories.
- Snow recognized importance, others less-so
- Snow lacked statistical tools for definitive conclusion
- **Time-pattern (epidemic curve)** also new, also separates theories

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Summarizing Predictions vs Evidence

Lakatos's *Sophisticated Falsification* requires:

- 1 T' has excess empirical content (novel facts)
- 2 T' explains previous success of T
- 3 Some of the excess content of T' is corroborated

Fecal-oral satisfies all:

- New facts ("no other factors" 5c-f, "epidemic curve" 8&9, even "contagion" 2b) corroborated
- For airborne, auxiliary water is *degenerating* (ad hoc) hypothesis – produces no new facts

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Summarizing Predictions vs Evidence

Strong argument that fecal-oral was the better theory

- Predicted novel facts, that were corroborated

But still (in 1856) rational reasons to be cautious about fecal-oral

- *Mechanism* not well understood – Or rather not well-recognized
 - By 1866, Farr had visited and recognized Pacini's identification of *vibrio cholerae*
- Could not test for and trace “cholera poison”